Pathologic Quiz Case
An Adrenal Mass in a 54-Year-Old Man

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A 54-year-old, 112-kg black man presented with primary hyperaldosteronism. His medical history was significant for multiple myeloma diagnosed 10 years earlier, difficult-to-control hypertension, glaucoma, hypothyroidism diagnosed 1.5 years earlier, and an intramuscular lipoma. His current medications included methadone 10 mg by mouth twice daily (PO BID), celecoxib 100 mg PO BID, gabapentin 300 mg PO once daily (QD), minoxidil 10 mg PO QD, atenolol 50 mg PO QD, levothyroxine 0.25 mg PO QD, and spironolactone 100 mg PO QD. On admission, he had a sodium (Na\(^+\)) concentration of 135 mEq/L, a potassium (K\(^+\)) concentration of 4.0 mEq/L, a chloride (Cl\(^-\)) concentration of 94 mEq/L, and a bicarbonate (HCO\(_3^-\)) concentration of 27 mEq/L. A computed tomography scan of the abdomen showed a modestly enlarged right adrenal gland with a 1.2-cm nodule. The patient underwent a right laparoscopic adrenalectomy. Intraoperative findings were severe perihepatitis and a right adrenal cortical nodule.

Grossly, the specimen was an 11.5-g, 7.0 × 3.0 × 2.0-cm adrenal gland containing a 1.5 × 1.3 × 1.2-cm well-circumscribed golden yellow nodule. Histologically, under low-power magnification, the lesion was well-circumscribed with an alveolar or nesting pattern separated by fine microvasculature (Figure 1). On higher power magnification, populations of cells with variable morphology were identified. The more common populations were large cells that had a pale-staining cytoplasm and were admixed with smaller cells with less abundant but more eosinophilic cytoplasm. The most striking feature was the numerous concentric eosinophilic cytoplasmic inclusions throughout the lesion (Figure 2).

What is your diagnosis?
Pathologic Diagnosis: Adrenocortical Adenoma With Spironolactone Bodies

Primary hyperaldosteronism is usually caused by an adrenocortical adenoma (65%–88%) and, rarely, by an adrenocortical carcinoma. Patients are usually women in their fourth to fifth decades of life. Patients present with hypertension, averaging 200 mm Hg systolic and 120 mm Hg diastolic; weakness; hypokalemia; and lassitude. The diagnosis is made by clinical evaluations and laboratory criteria. Initial presentation will show hypokalemia with a potassium concentration of less than 4 mEq/L, a normal serum sodium concentration, low plasma renin activity, and elevated plasma and/or urine aldosterone concentrations. Once primary hyperaldosteronism is suspected, a computed tomography scan will differentiate between hyperplasia and an adenoma. The treatment of choice for a primary hyperaldosteronism due to a single nodule is a unilateral adrenalectomy, most commonly via laparoscopy.

Grossly, the tumor is a yellow-orange, solitary, well-circumscribed nodule averaging 1.5 cm in diameter and is unencapsulated or incompletely encapsulated. Histologically, the tumor may have variable cell morphology, with the most common population of cells being large cells with pale cytoplasm and vesicular chromatin resembling cells from the zona fasciculata. Other cell populations may include smaller cells with eosinophilic cytoplasm and more condensed chromatin resembling cells from the zona reticularis.

Patients treated with the aldosterone agonist spironolactone may have spironolactone bodies. Spironolactone is a potassium-sparing diuretic that acts on the distal tubules and collecting ducts. It competitively binds the aldosterone receptor, thus blocking the reabsorption of sodium. Spironolactone bodies are concentric laminated eosinophilic inclusions within the endoplasmic reticulum of the tumor cells or the adjacent zona glomerulosa. They range in size from 2 to 12 μm, are periodic acid–Schiff positive and diastase resistant, and stain dark blue with Luxol fast blue. They are seen while the patient is taking the medication and have no apparent correlation with dosage or duration of treatment. However, they are unlikely to be present when the medication is stopped for more than a day. Their origin is not known, but they react positively for aldosterone by immunohistochemistry.

Although a rare neoplasm, a differential diagnosis of an adrenocortical carcinoma must be considered. It has a bimodal peak in age incidence occurring in the first decade of life and in the forth to fifth decades of life. Unlike the adrenocortical adenoma, the average weight of the adrenal gland can range from 510 to 1200 g and measure from 12.0 to 16.0 cm. There are usually areas of focal hemorrhage and necrosis. The most common histologic pattern is that of broad anastomosing trabeculae with sinusoids. Nuclear pleomorphism, high mitotic activity, and atypical mitoses also help differentiate adrenocortical carcinoma from adrenocortical adenoma.

References